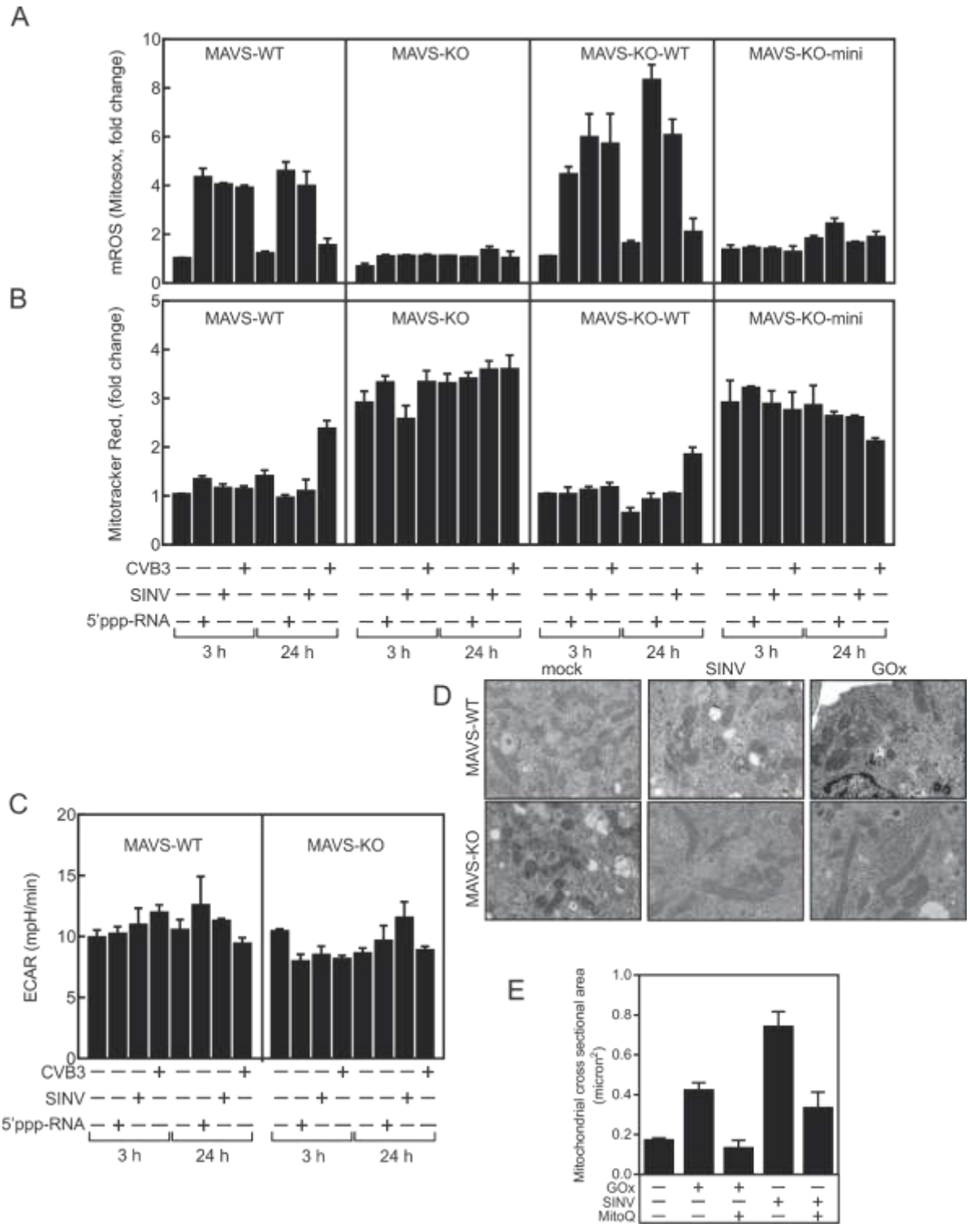
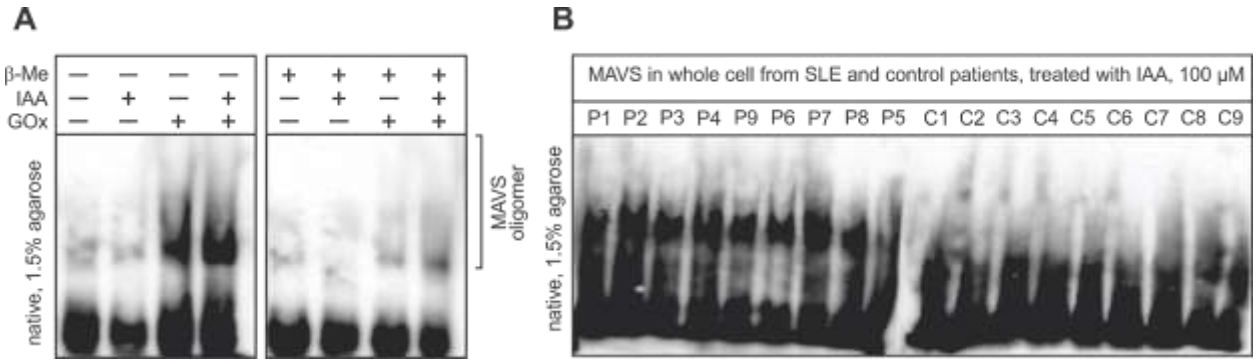
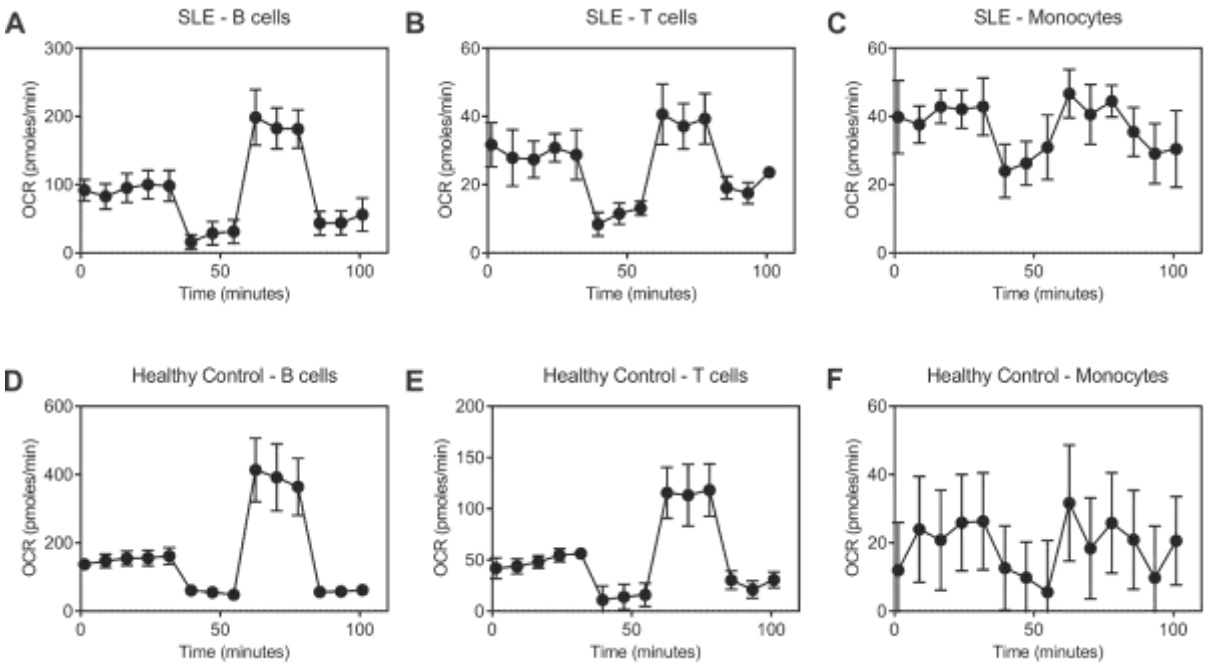
SUPPLEMENTARY MATERIALS



Supplementary Figure 1. Measurement of mROS, mitochondrial mass and mitochondrial phenotype. (A) Generation of mROS in WT, MAVS-deficient, reconstituted, or minimal-MAVS (truncated at position Gln148) expressing MEF in response to infection with CVB3 or SINV, or transfection with 5′-ppp–RNA, was quantified by FACS using 50 nM MitoSox Red. (B) Flow cytometric quantification of total mitochondrial mass in these cells was achieved by MitoTracker DeepRed. (C) The basal extracellular acidification rate (ECAR), a readout of lactate production by glycolysis, was measured by Seahorse Flux Analyzer. (D) The ultrastructural morphology of mitochondria in WT and MAVS-deficient MEFs following infection was observed by transmission electron microscopy. (E) The dimensions of WT and MAVS-deficient mitochondria were calculated from representative electron microscopic images, and the percentage of elongated mitochondria was calculated from 5 micrographs.



Supplementary Figure 2. MAVS oligomers are not artificially formed during sample processing, homogenization or lysis. (A) Freshly prepared PBMC were treated with GOx or infected with SINV, and subsequently homogenized in buffer containing 100 µM iodoacetamide. (B). PBMC of SLE patients (n=9) and healthy, sex-and age matched control donors (n=9) were isolated by Ficoll gradient centrifugation as described, and cells and corresponding plasma samples were supplemented with 100 µM iodoacetamide before analysis.



Supplementary Figure 3. Analysis of mitochondrial respiration in PBMC subpopulations. OCR in B cells (A), T cells (B) and monocytes (C) isolated from patients with SLE was measured following the sequential addition of oligomycin, FCCP, and a combination of antimycin A and rotenone. B cells, T cells and monocytes (D, E, and F, respectively) from healthy individuals served as controls. Shown are representative data of three independent experiments.